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Cognitive Effects of Aluminum Exposure in Cement Factory Workers: A Mini-Mental State Examination (MMSE) Assessment*

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Abstract

Background: *Aluminum (Al) is a widely encountered heavy metal with known neurotoxic effects. Occupational exposure, particularly in industrial settings, may impair cognitive functions. This study aimed to evaluate the relationship between Al exposure and cognitive function.* **Methods:** *A retrospective study was conducted at Gazi University Faculty of Medicine Hospital, Occupational Diseases Outpatient Clinic, between December 5, 2024, and January 5, 2025. The exposed group consisted of 20 male cement factory workers with elevated urinary Al expressed as a function of creatinine, and the control group included 40 age-matched males without occupational Al exposure (1:2 matching). Cognitive status was assessed using the Mini Mental State Examination (MMSE). Urinary Al levels were measured by inductively coupled plasma mass spectrometry (ICP-MS). Statistical analyses were performed with SPSS 29.0.* **Results:** *The mean MMSE score was significantly lower in the exposed group compared with controls (24.3±3.7 vs. 28.5±2.3, p<0.001). Subscale scores for orientation, attention/calculation, recall, and language were also lower in exposed workers. All such workers had elevated aluminum (mean 42.3±21.4 mcg/g creatinine). Urinary Al was positively correlated with working duration (r=0.453, p=0.045) and negatively correlated with MMSE (r=-0.486, p=0.030) and orientation scores (r=-0.494, p=0.027).* **Conclusion:** *Workers occupationally exposed to aluminum exhibited significantly lower cognitive performance than non-exposed controls. Higher urinary Al levels were associated with poorer cognitive outcomes, suggesting neurotoxic effects of aluminum and underscoring the importance of preventive strategies and cognitive monitoring in exposed populations.*

1. Introduction

Aluminum (Al) is a metal found in nature combined with other elements such as silicon and oxygen, and obtained from aluminum-containing minerals [1]. Many people are exposed to aluminum through media and routes such as water, fruits and vegetables, processed foods, some medications and vaccines, and food-heating and storage devices (such as aluminum foil) [2]. Occupational exposure occurs during the production, processing, welding, and recycling of aluminum. It is stated that occupational

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exposure to aluminum results from workers' contact, especially through inhalation [4]. The absorption of aluminum is affected by chemical form, particle size, and the use of dietary chelators (such as citric or lactic acid) [3]. Blood/serum and urine analyses can be performed to monitor aluminum exposure [5].

Studies have shown that aluminum has adverse effects on many systems, including the respiratory, cardiovascular, and gastrointestinal systems [6, 7]. Monographs published by IARC have identified a relationship between aluminum smelting plants and bladder cancer [4]. The nervous system is the most sensitive system to aluminum toxicity [8, 9]. Al exposure is thought to be associated with Alzheimer's, Parkinson's, Multiple Sclerosis, and many other neurological diseases [10-12]. In the meta-analysis conducted by Soleimani et al., it was determined that aluminum exposure increased the risk of Alzheimer's disease by 2.451 times (CI: 0.569-4.334, p=0.011) [13]. When Alzheimer's patients were compared with control patients, aluminum levels were higher in the cerebrospinal fluid, serum, and brain tissue of Alzheimer's patients [14].

It has been reported that aluminum exacerbates oxidative damage due to its pro-oxidant properties and may therefore be a risk factor in ALS [15]. In a review by Killin et al., it was reported that increased aluminum levels in water increase the risk of dementia [16]. Neurotoxic effects of aluminum are explained through various biochemical pathways. As a prooxidant, it can cause oxidative stress and lead to the production of free oxygen molecules. It can also cause mitochondrial dysfunction [17]. As a cholinotoxic agent, it can alter acetylcholinesterase activity [18].

Additionally, considering the effects of aluminum on cognitive functions, workers with occupational exposure were found to have poorer performance in attention, reaction time, and working memory. It has been determined that elevated plasma aluminum levels can predict declines in cognitive performance. In a study of shift workers at an aluminum smelting plant in Norway, it was reported that alertness levels were lower after night shifts than after 3-4 consecutive day shifts [20]. In a cross-sectional study, aluminum levels were higher in cognitively impaired workers than in controls, and gray matter volumes in the left caudate and bilateral hippocampus were lower and positively correlated with aluminum levels [9]. In a study of workers in an aluminum factory, the risk of cognitive impairment increased 2.24-fold when plasma aluminum levels were high compared with low [8].

Based on the literature, our study aimed to evaluate the effects of aluminum exposure on cognitive function. For this purpose, we aimed to determine the relationship between aluminum intoxication and cognitive status assessment in patients referred to us from the cement production industry with suspicion of aluminum intoxication and in the control group.

2. Methods

This study was designed as a retrospective cross-sectional study. The study population consisted of workers who attended the Occupational Diseases Outpatient Clinic of Gazi University Faculty of Medicine between December 5, 2024, and January 5, 2025.

Patients referred by occupational physicians or other physicians with suspected occupational diseases are evaluated at our outpatient clinic. During this period, all clinical and laboratory evaluations were performed as part of routine occupational health assessments, and the data were recorded in institutional medical records. No data were collected specifically for research purposes at that time. After obtaining ethical approval from the Gazi University Rectorate Ethics Committee (No: 2025-1337; date: 29/07/2025), these existing records were retrospectively reviewed and analyzed.

2.1. Occupational Exposure

The cement factory examined in this study produces ready-mixed cement through the stages of raw material preparation, mixing, firing, and grinding. The cement produced in the factory contains limestone and silica; hydraulic hardening occurs through calcium silicates and aluminates. Raw materials, including limestone, silica, and clay-based minerals, contain aluminum-bearing compounds that are essential components of Portland cement chemistry. Workers in the exposed group in this study

are potentially exposed to aluminum-containing cement dust, especially during raw material handling, mixing, kiln operation, and clinker grinding. Occupational exposure occurs mainly through inhalation of cement dust containing aluminum compounds. According to available records, workers were not consistently able to use personal protective masks, resulting in continuous inhalation exposure to cement dust containing aluminum during their shifts.

2.2. Population

2.2.1. Exposed Group

Twenty male cement factory workers with elevated urinary aluminum levels or aluminum/creatinine ratios were identified from medical records. To ensure that cognitive impairment was specifically attributable to aluminum exposure and not to polymetallic contamination or other confounders, inclusion criteria required that participants have normal levels of blood lead (<8.5 μg/dL), and urinary chromium (<5 μg/L), mercury (<9 μg/L), and manganese (<18.3 μg/L). Additionally, serum vitamin B12 levels had to be within the normal range (197-771 pg/mL). All cement factory workers who agreed to participate and met these criteria were included in the study.

2.2.2. Control Group

Forty age-matched male individuals without aluminum exposure were identified from medical records and met the same heavy metal and vitamin B12 criteria. Exposed – control matching was performed 1:2.

2.2.3. Exclusion Criteria

Participants with elevated blood lead (>8.5 μg/dL), serum chromium (>5 μg/L), blood mercury (>9 μg/L), or blood manganese (>18.3 μg/L) levels, decreased B12 (<197 pg/mL) or history of physician-diagnosed epilepsy, dementia, learning disabilities, and depression; as well as those who declined to participate, were excluded from the study. These thresholds correspond to the reference intervals provided by the accredited clinical laboratory performing the analyses and were used only as screening criteria to exclude participants with markedly elevated levels of other heavy metals.

All clinical evaluations and laboratory measurements had been performed as part of routine clinical practice and were obtained from medical records. Urine and blood samples had been collected at the end of 8-hour work shifts as part of routine occupational exposure assessment. Sample analysis was performed using an ICP-MS device. Urine samples were collected in sterile containers at the end of the shift. Heavy metal levels were analyzed using inductively coupled plasma mass spectrometry (ICP-MS, Agilent 7700X, USA). The argon used in the plasma torch had a purity of over 99.999% and was supplied by HABAS (Kocaeli, Turkey). In the analyses, 65% Suprapur nitric acid (Merck, Darmstadt, Germany) and an aqueous multi-element standard solution (High-Purity Standards, Charleston, USA) were used. Calibration was performed with an aqueous multi-element standard solution. The method was validated by analyzing certified reference materials (Seronorm Trace Elements, Billingstad, Norway). Urine aluminum levels were determined by the ICP-MS method. According to international standards, reference values for aluminum in spot urine are <35 μg/L or ≤14 mcg/g creatinine in spot urine samples [21].

The Mini-Mental State Examination (MMSE) was used to assess participants' cognitive status. Developed by Folstein et al., the MMSE consists of eleven questions and is scored out of 30. It has five subscales: orientation (10 points), registration (3 points), attention and calculation (5 points), recall (3 points), and language and copying (9 points). Orientation assesses an individual's awareness of temporal and spatial context, while immediate memory is assessed by recording and repeating presented words. Attention and calculation measure concentration and working memory using tasks such as serial subtraction or spelling. Recall examines short-term memory by testing delayed recall of previously presented words. Language and copy assess a range of linguistic functions, including naming, repetition, comprehension, reading, and writing,

as well as the ability to accurately reproduce a simple geometric shape. The Turkish translation was made by Güngen et al. [22, 23].

Diagnosis had been established based on clinical findings, laboratory measurements, and Mini Mental State Examination (MMSE) results, as documented in medical records. This process was conducted during a multidisciplinary team meeting involving psychologists, neurologists, and occupational disease specialists.

2.3. Statistical Analysis

Descriptive statistics for the study are presented as numbers, percentages, means, and standard deviations. Normality of continuous variables was assessed using the Shapiro-Wilk test. Chi-square tests, Mann-Whitney U tests, and Spearman's rank correlation were used to assess relationships between groups. Multiple linear regression analysis (enter method) was performed with MMSE score as the dependent variable, and urinary aluminum, aluminum/creatinine ratio, age, education group, and smoking status as independent variables. Statistical analyses were performed using SPSS 29.0. P<0.05 was considered significant.

3. Results

Our study was conducted with 20 patients with aluminum exposure and 40 controls. The mean age of the exposed subjects was 42.99±8.3 years, and the control group was 39.7±8.9 years (p=0.174). The exposed group had longer working experience (p=0.013). Seventeen (85.0%) of the exposed group and 30 (75%) of the control group were married (p=0.375), while 32 (80.0%) of the control group and 11 (55.0%) of the exposed subjects had a high school education or higher (p=0.037). The smoking rate was higher in workers with Al exposure than in those without (p=0.013). However, the number of cigarettes smoked was similar in smokers (p=0.367). In the exposed group, aluminum concentrations ranged from 13.59 to 67.09 μg/L (mean 30.1±14.9 μg/L), while aluminum values were above the normal range in three participants. The corresponding values expressed as a function of creatinine ranged from 20.98 to 100.52 mcg/g (mean 42.3±21.4 mcg/g); thus, all values expressed as a function of creatinine were above the upper reference limit. The corresponding values of the control group were 1.87 ± 0.84 μg/L and 2.05 ± 1.01 μg/g creatinine, respectively, both p<0.001 vs. exposed workers (Table 1).

Table 1. Characteristics of the study group.

	Exposed group		Control		
	Mean±SD/n	**Percentage (%)**	**Mean±SD/n**	**Percentage (%)**	**p**
Age	42.9±8.3		39.7±8.9		0.174*
Working Time	22.4±8.6		16.6±9.8		0.013*
Marital Status					0.375
Single	3	15.0	10	25.0	
Married	17	85.0	30	75.0	
Education Level					**0.037**
Primary and Secondary School	9	45.0	8	20.0	
High School and Above	11	55.0	32	80.0	
Smoking Status					**0.013**
No	3	15.0	19	47.5	
Yes	17	85.0	21	52.5	
Cigarettes (Packs/year)	18.6±9.6		26.5±20.5		0.367*
Urinary Al (μg/L)	30.1±14.9		1.87±0.84		<0.001*
Urinary Al (mcg/g creatinine)	42.3±21.4		2.05±1.01		<0.001*

** Mann Whitney U Test, SD: Standard Deviation.*

The MMSE score was 24.3±3.7 in workers with aluminum exposure, while it was 28.5±2.3 in those without (p<0.001). Furthermore, scores in the orientation, attention, calculation, recall, and language subscales were also lower in the exposed group (Table 2).

There was a positive correlation between the urinary aluminum and total working hours (r=0.453, p=0.045) and a negative correlation with both the orientation score (r=-0.494, p=0.027) and MMSE (r=0.486, p=0.030). However, such correlations were significant only when urinary Al was expressed as a function of creatinine, whereas no correlation was observed between raw urinary Al (unadjusted) and MMSE scores (Table 3). The latter were also sensitive to education, with those with primary and

Table 2. Cognitive assessment of the study group.

	Exposed group		Control		
	Mean±SD	Median (min-max)	Mean±SD	Median (min-max)	p
Orientation	8.8±1.4	9.0(5-10)	10.0±0.0	10.0 (10-10)	<0.001
Registration	3.0±0.0	3.0(3-3)	2.9±0.3	3.0 (1-3)	0.313
Attention and calculation	1.9±2.1	1.0 (0-5)	4.0±1.6	5.0 (0-5)	<0.001
Recall	2.4±0.9	3.0 (0-3)	2.8±0.5	3.0 (1-3)	0.003
Language and copying	8.3±1.0	9.0 (6-9)	8.8±0.5	9.0 (7-9)	0.032
Total score (MMSE)	24.3±3.7	24.0(17-30)	28.5±2.3	30.0 (23-30)	<0.001

Mann Whitney U Test, SD: Standard Deviation.

Table 3. Correlation of factors affecting MMSE scores.

		Aluminum	Aluminum/Creatinine	Working Hours
MMSE	r	0.289	-0.486	-0.197
	p	0.216	**0.030**	0.132
Orientation	r	0.199	-0.494	-0.208
	p	0.400	**0.027**	0.111
Attention and calculation	r	0.271	-0.420	-0.170
	p	0.248	0.065	0.195
Recall	r	0.099	-0.247	-0.236
	p	0.679	0.294	0.069
Language and copying	r	0.206	-0.057	-0.225
	p	0.384	0.813	0.046
Working Hours	r	0.054	0.453	
	P	0.823	**0.045**	1

**Spearman Correlation.*

Table 4. Predictors of MMSE Score in Multivariate Analysis.

	B	SE	β	t	p	95% CI
Aluminum (μg/L)	−0.015	0.021	−0.067	−0.689	0.494	−0.057 – -0.028
Aluminum (μg/g creatinine)	−0.050	0.015	−0.332	−3.260	0.002	−0.081 – −0.019
Age (Years)	0.001	0.030	0.002	0.023	0.981	−0.059 – 0.061
Education Group	2.292	0.317	0.604	7.240	<0.001	1.657 – 2.927
Smoking Status	-0.659	0.544	-0.093	-1.211	0.213	-1.751 – 0.432
Constant	20.582	1.906		10.798	<0.001	16.761 – 24.403

B: unstandardized coefficient, SE: Standard Error, β: standardized coefficient. CI: Confidence Interval.

secondary school degrees showing lower scores than those with high school education or above (21.3±2.3 vs. 26.7±2.7, p<0.001). There were no correlations between age and MMSE, orientation, memory, attention and calculation, recall, and language.

Multiple linear regression analysis was conducted to identify factors associated with MMSE. Urinary Al, when expressed as a function of creatinine, was negatively and independently associated with MMSE; in contrast, the education group showed a strong positive association with the mental score. Age, smoking status, and urinary aluminum concentration alone were not significantly associated with MMSE (Table 4).

4. Discussion

To our knowledge, this is the first study comparing the cognitive status of participants working in the cement industry in Türkiye who have high aluminum levels with that of those not exposed to aluminum. Our study demonstrated cognitive impairment in workers with Al exposure. There are studies in the literature evaluating the occurrence of cognitive impairment due to environmental and occupational exposure to Al. In the meta-analysis, urine aluminum levels in controls averaged 9.61 μg/L (SD = 4.95) and 11.32 μg/g creatinine (SD = 8.13), compared with exposed workers at 87.20 μg/L (SD = 82.92) and 54 μg/g (SD = 38.28), respectively [19]. In this study, cement workers showed markedly higher urinary aluminum and aluminum-to-creatinine ratios than controls, indicating a clear exposure gradient consistent with occupational aluminum exposure. Although environmental air monitoring data were unavailable, biological monitoring demonstrated a clear exposure gradient between cement workers and controls, supporting the conclusion of occupational aluminum exposure. The results of the meta-analysis show decreased performance in processing speed, working memory, attention, and reaction time among workers occupationally exposed to aluminum compared with controls.

In a study by Zhang et al. of aluminum mine workers, the MMSE score was 21.34 among workers with aluminum exposure and 22.95 among those without. The MMSE score was lower in those with aluminum exposure. Furthermore, in an analysis adjusted for age, gender, and education level, the risk of cognitive impairment was reported to be 6.7 times higher in those with aluminum exposure [24]. In the study by Xing et al., the MMSE score was reported as 27.93 ± 1.91 in the Al-exposed group and 28.62 ± 1.25 in the non-exposed group. The score was lower in the exposed group [25]. In the study by Lu et al., the MMSE score in workers exposed to aluminum was reported as 26.13±2.57, which was lower than that of the control group [26]. Additionally, some studies have found that MMSE scores are lower among workers with aluminum exposure than among those without [27, 28]. In our study, the MMSE score was 24.3±3.7 in workers with occupational Al exposure and 28.5±2.3 in non-exposed workers, which is consistent with findings reported in the literature. In addition, aluminum normalized to creatinine was elevated in all exposed participants. This observation suggests that creatinine-adjusted urinary aluminum may represent a more

informative indicator of occupational aluminum exposure when spot urine samples are used, as it helps account for inter-individual variability in urine dilution. Overall, these findings suggest that increased aluminum exposure may be associated with lower cognitive performance.

In evaluating MMSE subscales, Xing et al. found that orientation, recall, attention, calculation, and language scores were similar between the aluminum- and non-aluminum-exposed groups. Visual ability scores were significantly higher in the control group [25]. In the study by Lu et al., scores for orientation, recall, calculation ability, and language skills were lower among workers exposed to aluminum than in the control group [26]. In Yang et al.'s study, recall, calculation ability, and language skill scores were also found to be lower in the group with the highest aluminum level than in the group with the lowest [29]. Similarly, in our study, orientation, recall, attention, calculation, and language scores were lower among workers exposed to aluminum. Furthermore, orientation scores were negatively affected by an increased aluminum-to-creatinine ratio. These results support the negative consequences of aluminum exposure on cognitive functions.

A study by Qui et al. of aluminum factory workers reported lower MMSE scores among workers with higher plasma aluminum levels than among those with lower levels [30]. In a study by Lu et al., which followed workers at an aluminum factory for 2 years, MMSE scores decreased as workers' aluminum levels increased during the follow-up [31]. Additionally, many studies have found a negative correlation between blood plasma aluminum levels and MMSE scores [27-29, 32-34]. When studies evaluating blood aluminum level and MMSE score are evaluated, Pan et al reported an increase of -0.005 times (p=0.001), Abdala et al reported an increase of -0.348 times (p=0.017 CI:-0.632,-0.065), and Zhang et al. reported an increase of -0.630 times (p<0.001) [32, 34, 35]. Contrary to these studies, some studies reported no relationship between aluminum levels and MMSE scores [36-38]. In our study, there was a negative correlation between increased aluminum-to-creatinine ratio and MMSE scores. This supports the notion that increased aluminum exposure will lead to a decline in cognitive function.

Individual factors such as age, education level, and length of service also influence MMSE scores. A study by Pan et al. reported that MMSE scores were negatively affected by age (-0.061, p=0.001) and length of service (-0.058, p=0.001), while education level had no effect [32]. Lu et al. reported that age and education level affect [26]. Abdalla et al. determined that the study period increased the score by -0.044 times (p=0.032, CI:-0.084 to -0.004) [34]. Shang et al. reported that the year was positively correlated in their study [27]. Our study also found that higher education levels served as a protective factor for cognitive function, while working hours had a negative impact. Higher education levels are known to protect cognitive function.

5. Conclusion

In conclusion, this study shows a significant and independent association between aluminum exposure and cognitive performance, as measured by the Mini-Mental State Examination (MMSE), when urinary aluminum levels are expressed as a function of creatinine. Although no correlation was observed when aluminum concentrations in spot urine were analyzed, a significant association was detected when aluminum normalized to creatinine in spot urine was used, highlighting the importance of correcting for urine dilution in biomonitoring studies. Despite the known limitations of the MMSE, the persistence of this link after controlling for key confounding factors suggests that increased aluminum exposure may negatively affect cognitive function beyond the protective effects of cognitive reserve. Consistent with aluminum's known neurotoxic properties, these findings support its potential role as an occupational factor contributing to cognitive impairment and emphasize the importance of considering aluminum exposure among at-risk workers. Additionally, given its low cost, reproducibility, and ease of use, the MMSE could be a practical tool for occupational physicians to monitor cognitive decline among exposed workers.

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Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Ethics Committee

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. Written informed consent has been obtained from the patient(s) to publish this paper.

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